DYSPEPSIA MANAGEMENT IN A RESOURCE POOR SETTING

A.C. Jemilohun¹ and J.O. Fadare²

- 1. Department of Medicine, Ladoke Akintola University of Technology, Osogbo, Osun State, Nigeria.
- 2. Department of Clinical Pharmacology, Obafemi Awolowo University, Ile-Ife, Osun State, Nigeria.

Correspondence:

Dr. A.C. Jemilohun

Department of Medicine, College of Health Sciences, Ladoke Akintola University of Technology, Osogbo, Osun State, Nigeria.

E-mail: chrislohun2010@hotmail.com.

ABSTRACT

Background: Dyspepsia has a significant impact on the quality of life of the sufferer, and results in enormous societal costs, either due to direct medical costs for physician visits, diagnostic tests, medications, or indirect costs from absenteeism or reduced productivity at work. It is therefore important to explore the management options available, especially in a resource poor setting like Nigeria, in the light of the foregoing.

Methods: Extensive internet literature search was made through Google scholar, Pubmed and HINARI. Keywords employed were dyspepsia, prevalence and management.

Result: Several approaches proposed for the management of a newly diagnosed patient with dyspepsia include: empirical trial of acid suppression for 4-8 weeks in regions with low prevalence of *H. pylori*; the "test and treat" approach for *H. pylori* infection using a validated non-invasive test and; initial use of upper gastrointestinal endoscopy to determine the nature of the disease before treatment in patients with alarm symptoms and those who are more than 45 years. Helicobacter pylori eradication therapy without initial diagnostic testing can be used as the last result in resource poor regions of the word where diagnostic tests for *H. pylori* are not available or not cost-effective.

Conclusion: Considering the high cost of upper gastrointestinal endoscopy and the high prevalence of *H. pylori* infection in developing countries like Nigeria, it seems reasonable that the 'test and treat' method will be of immense usefulness in population sub-group who are less than 45 years without alarm symptoms, while those with alarm symptoms and those with onset of symptoms after 45 years will require initial upper gastrointestinal endoscopy.

Keywords: Dyspepsia, Classification, Pathophysiology and Management.

INTRODUCTION

Dyspepsia is defined as chronic or recurrent central upper abdominal pain or discomfort which is referable to the upper gastrointestinal tract^{1,2}. It is usually associated with intake of food or hunger. Discomfort according to the Rome Working Teams refers to a subjective, negative feeling that does not reach the level of pain according to the patient. This can incorporate a variety of symptoms such as upper abdominal fullness, early satiety, bloating, belching or nausea^{1,2}.

Dyspepsia is a common presentation in clinical practice worldwide^{1, 2}. It has a prevalence of between 20% and 40% in the adult population ^{3, 4}. In a study carried out among the British population it was found to be 38%⁵. It is estimated to account for 2% to 5% of primary care office visits and 30% of consultations by

Gastroenterologists^{6, 7}. A prevalence of 26% to 45% was found in some parts of Nigeria^{8, 9}. Dyspepsia has a significant impact on quality of life¹⁰, and results in enormous societal costs, either due to direct medical costs for physician visits, diagnostic tests, medications, or indirect costs from absenteeism or reduced productivity at work^{11, 12}. It is therefore, important to explore the management options available in the light of the foregoing.

CLASSIFICATION OF DYSPEPSIA

Dyspepsia can be broadly classified into two major groups. These include organic dyspepsia and functional dyspepsia.

Organic dyspepsia: This is dyspepsia that results from a structural or anatomical lesion. These structural lesions include chronic gastritis, duodenitis, gastric and duodenal erosions, gastric and duodenal ulcers, gastric adenocarcinoma and mucosal associated lymphoid tissue (MALT) lymphoma.^{13,14} *Helicobacter pylori* infection has been noted to be associated with most of the disease entities presenting as dyspepsia.^{13,14} The particular end result of *H. pylori* infection is determined by a complex interaction between bacterial, host and other environmental factors.¹³ A detailed description of this interaction is beyond the scope of this review.

Functional dyspepsia: This is dyspepsia in which there is no evidence of organic disease that can adequately explain the symptoms. It is also known as idiopathic or non-ulcer dyspepsia, and is often a diagnosis of exclusion. Many patients with functional dyspepsia (FD) have multiple somatic complaints, as well as symptoms of anxiety and depression. It is further subdivided clinically into ulcer-like, reflux-like, dysmotility-like, and non-specific dyspepsia. This sub-grouping, however, has not been found to be of much practical value in identifying the underlying cause of dyspepsia as the symptoms overlap considerably.

The pathophysiology of functional dyspepsia is poorly understood. There is symptom overlap with those of other functional gastrointestinal disorders, such as functional heartburn, irritable bowel syndrome (IBS), and non-cardiac chest pain. 17 Like other functional gastrointestinal disorders, FD is best understood in the context of the bio-psychosocial model of illness in which symptoms arise out of a complex interaction between abnormal gastrointestinal physiology and psychosocial factors that affect how a person perceives, interprets, and responds to the altered gastrointestinal physiology.¹⁸ Several pathophysiological mechanisms that have been suggested as playing a part in its development include delayed gastric emptying, 18,19 impaired gastric accommodation, 20,21 myoelectric abnormalities, 22,23 altered antro-duodeno-jejunal motility²⁴, visceral hypersensitivity,²⁵ altered vagal function,26 altered duodenal sensitivity to lipids or acid, 27, 28 and psychological disorders. 29,30

MANAGEMENT

Several approaches that have been proposed for the management of a newly diagnosed patient with dyspepsia include:^{1,31}

- 1. Empirical trial of acid suppression with antisecretory drugs like proton pump inhibitor (PPI) or Histamine 2 receptor blocker for 4-8 weeks
- 2. The "test and treat" approach for *H. pylori* infection using a validated non-invasive test and a trial of

- gastric acid suppression if eradication is successful but symptoms do not resolve, and
- 3. Initial upper gastrointestinal endoscopy (UGE) to determine the nature of the disease.

Empirical trial of acid suppression

This approach is recommended in populations with low prevalence of *H. Pylori* infection (<10%).^{1,32} It is done using antisecretory drugs like proton pump inhibitor (PPI) or Histamine 2 receptor blocker for 4-8 weeks. If there is no amelioration of symptoms within 2-4 weeks of commencement of treatment, it is recommended that drug class be changed. Generally, PPIs have been found to be more effective than the H2RBs.

Although this approach is cheap, a major drawback to its use is the generally high prevalence of *H. pylori* infection in regions of the world with poor socioeconomic condition.

'Test and treat' method

With the burden of evidence implicating H. pylori in the aetiology of different diseases manifesting clinically as dyspepsia, it will be appropriate for all patients with dyspepsia who are positive for H. pylori to undergo H. pylori eradication therapy. 1,5,9 More so, that H. pylori eradication has been associated with significant reduction in rate of recurrence of peptic ulcer disease and cure of MALT. 13 When there are no 'alarm symptoms' the 'test and treat' method using multidrug therapy for H. pylori eradication is a rational approach, especially in populations with a moderate to high prevalence of H. pylori infection ($\geq 10\%$) followed by a course of empirical antisecretory therapy in patients who fail to respond or relapse rapidly on stopping H. pylori eradication therapy. 1,33,34

The urea breath test and the stool antigen test are the recommended tests in non-invasive diagnosis of *H. Pylori* because of their high diagnostic accuracy.^{35, 36} Serological tests are not recommended because of their low discriminatory power between old and current infections. They cannot also be used to ascertain cure of infection.

One major drawback to this approach in developing countries like Nigeria is the rarity of these non-invasive diagnostic tests of choice.

Initial upper gastrointestinal endoscopy

There is controversy as to when UGE should be done considering the cost and the risks involved. Nevertheless, UGE is clearly indicated when a patient with dyspepsia presents with any of the following features: Presentation with a first episode of dyspepsia

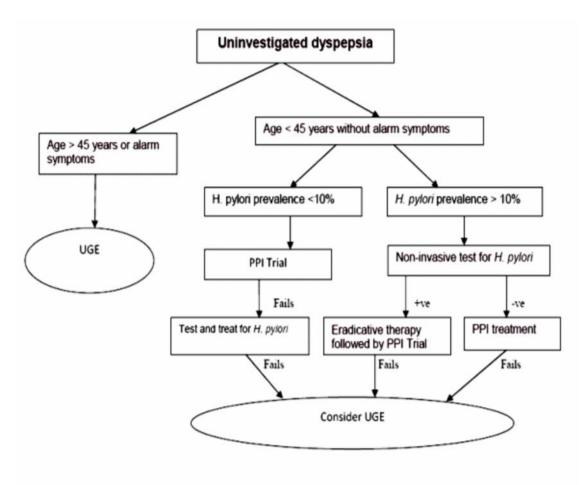


Figure 1: Algorithm for the management of uninvestigated dyspepsia

Adapted from: Talley NJ, Vakil N. Guidelines for the management of dyspepsia. Am J Gastroenterol. 2005; 100 (10):2324-2337

at >45 years of age (because of risk of malignancy), failure to respond to empirical anti-secretory therapy, and presence of alarm symptoms. 1, 32

Alarm symptoms include anorexia, weight loss, odynophagia, dysphagia, persistent vomiting, haematemesis, melaena, anaemia, unexplained weight loss (>10% body weight), a family history of gastrointestinal cancer, previous esophagogastric malignancy, lymphadenopathy, or an abdominal mass.¹

A careful history-taking, thorough physical examination and investigations such as abdominal ultrasound scan, barium studies, computer tomography and magnetic resonance imaging may be required for further characterization of disease in those who have alarm symptoms.

For younger patients who do not have alarm symptoms further diagnostic investigations are not usually required since upper gastrointestinal malignancy is rarely present in them, although the positive predictive value of alarm features remains very poor. ³⁷

Endoscopy may also be required to reassure patients who are worried that a malignant condition may be responsible for their symptoms. However, repeat endoscopy is not recommended once a diagnosis of non-ulcer dyspepsia has been clearly established in such patients, unless a completely new set symptoms or alarm features develop.

Treatment of endoscopy-negative dyspepsia (functional dyspepsia)

Endoscopy-proven functional dyspepsia is also treated with initial antisecretory therapy and *H. pylori* eradication just as organic dyspepsia. Management challenge arises when these measures fail because no other measure has been found to have optimal efficacy¹.

Simple reassurance as regards the benign nature of the illness may go a long way to ameliorate patients' anxiety. Occasionally, a reconsideration of diagnosis may be needed in order not to miss other conditions that may mimic dyspepsia. Dietary therapy may help some individuals although it has no established efficacy. Simethicone, low-dose tricyclic antidepressants and antispasmodics have all been used but there are very limited data supporting their efficacy.¹

Limited studies support psychotherapy, hypnotherapy, and cognitive-behavioral therapy but they cannot be generally recommended for now.¹

Helicobacter pylori eradication therapy without initial diagnostic test

This approach is usually the last result in resource poor regions of the word where diagnostic tests for *H. pylori* are not readily available or diagnostic tests for the infection are not cost-effective. ³⁸ The decision to treat is based on the assumption that *H. pylori* infection is present in patients with symptoms of dyspepsia since the prevalence of *H. pylori* is generally high in such settings. ^{14, 38} It is therefore better to treat the infection empirically than to do nothing because of patients' inability to afford the cost of investigation, considering the immense benefits accruing to the dyspeptic patient following eradication of the organism.

The drawback to this approach, however, is that one cannot say with all certainty that the organism has been eradicated after treatment.

CONCLUSION

Considering the high cost of UGE and the high prevalence of *H. pylori* infection in developing countries like Nigeria, ^{14, 38} it seems reasonable that the 'test and treat' method using recommended non-invasive tests will be of immense usefulness in population sub-group who are less than 45 years of age without alarm symptoms, while those with alarm symptoms irrespective of age and those with onset of symptoms after 45 years will require initial upper gastrointestinal endoscopy.

It is highly desirable that the recommended non-invasive diagnostic tests for *H. Pylori*, in addition to the existing gastrointestinal endoscopy facilities, are made available by policy makers. This will go a long way to improve the quality of care of patients, save cost of care and reduce the burden on the already overburdened Endoscopists and facilities for UGE in such populations.

REFERENCES

- 1. **Talley NJ,** Vakil N. Guidelines for the management of dyspepsia. Am J Gastroenterol. 2005; 100 (10):2324-2337.
- 2. **Bytzer P,** Talley NJ. Dyspepsia. Ann Intern Med. 2001; 134:815-822.
- 3. **Arents NL**, Thijs JC, Kleibeuker JH. A rational approach to uninvestigated dyspepsia in primary

- care: review of the literature. Postgrad Med J. 2002; 78 (926):707-716.
- 4. **Logan R,** Delaney B. ABC of the upper gastrointestinal tract: implications of dyspepsia for the NHS. BMJ. 2001; 323 (7314):675-677.
- Jones R, Lydeard S. Prevalence of symptoms of dyspepsia in the community. BMJ. 1989; 298 (6665):30-32.
- 6. **Maconi G,** Tosetti C, Stanghellini V *et al.* Dyspeptic symptoms in primary care. An observational study in general practice. Eur J. Gastroenterol Hepatol. 2002; 14(9):985-990.
- 7. **Majumdar SR,** Soumerai SB, Farraye FA *et al.* Chronic acid-related disorders are common and underinvestigated. Am J Gastroenterol. 2003; 98 (11):2409-2414.
- 8. **Holcombe C,** Omotara BA, Padonu MK, Bassi AP. The prevalence of symptoms of dyspepsia in North- Eastern Nigeria: A random community based survey. Trop Geogr Med. 1991; 43: 209-214.
- 9. **Ihezue CH,** Oluwole FS, Onuminya JE, Okoronkwo MO. Dyspepsias among the highlanders of Nigeria: an epidemiological survey. Afr J Med Med Sci. 1996; 25 (1):23-29.
- El-Serag HB, Talley NJ. Health-related quality of life in functional dyspepsia. Aliment Pharmacol Ther. 2003; 18:387-393.
- 11. **Agreus L,** Borgquist L. The cost of gastrooesophageal reflux disease, dyspepsia and peptic ulcer disease in Sweden. Pharmacoeconomics. 2002; 20 (5):347-355.
- 12. **Moayyedi P,** Mason J. Clinical and economic consequences of dyspepsia in the community. Gut. 2002; 50:10-12.
- 13. **Suerbaum S,** Michetti P. Helicobacter pylori infection. N Engl J Med. 2002; 347 (15):1175-1186.
- Oluwasola AO, Ola SO, Saliu L, Solanke TF. Helicobacter pylori infection in South Nigerians: a serological study of dyspeptic patients and healthy individuals. West Afr J. Med. 2002; 21(2):138-141.
- 15. **Dickerson LM,** King DE. Evaluation and management of nonulcer dyspepsia. Am Fam Physician. 2004; 70 (1):107-114.
- 16. **Fisher RS**, Parkman HP. Management of nonulcer dyspepsia. N Engl J Med. 1998; 339(19): 1376-1381
- 17. **Drossman DA,** Li Z, Andruzzi E *et al.* U.S. householder survey of functional gastrointestinal disorders. Prevalence, sociodemography, and health impact. Dig Dis Sci. 1993; 38(9):1569-1580.
- 18. **Fischler B,** Tack J, De Gucht V *et al.* Heterogeneity of symptom pattern, psychosocial factors, and pathophysiological mechanisms in

- severe functional dyspepsia. Gastroenterology. 2003; 124(4):903-910.
- 19. **Talley NJ,** Verlinden M, Jones M. Can symptoms discriminate among those with delayed or normal gastric emptying in dysmotility-like dyspepsia? Am J Gastroenterol. 2001; 96 (5):1422-1428.
- 20. **Lee KJ,** Kindt S, Tack J. Pathophysiology of functional dyspepsia. Best Pract Res Clin. Gastroenterol. 2004; 18 (4):707-716.
- 21. **Timmons S,** Liston R, Moriarty KJ. Functional dyspepsia: motor abnormalities, sensory dysfunction, and therapeutic options. Am J Gastroenterol. 2004; 99(4):739-749.
- 22. van der Voort IR, Osmanoglou E, Seybold M *et al.* Electrogastrography as a diagnostic tool for delayed gastric emptying in functional dyspepsia and irritable bowel syndrome. Neurogastroenterol Motil. 2003; 15(5):467-473.
- 23. **Holmvall P,** Lindberg G. Electrogastrography before and after a high-caloric, liquid test meal in healthy volunteers and patients with severe functional dyspepsia. Scand J Gastroenterol. 2002; 37(10):1144-1148.
- 24. **Jebbink HJ,** vanBerge-Henegouwen GP, Akkermans LM, Smout AJ. Small intestinal motor abnormalities in patients with functional dyspepsia demonstrated by ambulatory manometry. Gut. 1996; 38(5):694-700.
- 25. **Tack J,** Caenepeel P, Fischler B *et al.* Symptoms associated with hypersensitivity to gastric distention in functional dyspepsia. Gastroenterology. 2001;121(3):526-535.
- 26. **Holtmann G,** Goebell H, Jockenhoevel F, Talley NJ. Altered vagal and intestinal mechanosensory function in chronic unexplained dyspepsia. Gut. 1998;42(4):501-506.
- 27. **Simren M,** Tack J. Functional dyspepsia: evaluation and treatment. Gastroenterol Clin North Am. 2003; 32 (2):577-599.
- 28. **Barbera R,** Feinle C, Read NW. Nutrient-specific modulation of gastric mechanosensitivity in patients with functional dyspepsia. Dig Dis Sci. 1995; 40(8):1636-1641.

- 29. **Koloski NA,** Talley NJ, Boyce PM. Predictors of health care seeking for irritable bowel syndrome and nonulcer dyspepsia: a critical review of the literature on symptom and psychosocial factors. Am J Gastroenterol. 2001; 96(5):1340-1349.
- 30. **Moayyedi P,** Deeks J, Talley NJ *et al.* An update of the Cochrane systematic review of *Helicobacter pylori* eradication therapy in non-ulcer dyspepsia: resolving the discrepancy between systematic reviews. Am J Gastroenterol. 2003; 98(12):2621-2626.
- 31. **Moayyedi P.** *Helicobacter pylori* test and treat strategy for young dyspeptic patients: new data. Gut. 2002; 50:47-50.
- 32. Endoscopy in the evaluation of dyspepsia. Health and Public Policy Committee, American College of Physicians. Ann Intern Med. 1985; 102 (2):266-269.
- 33. **Talley NJ,** Axon A, Bytzer P *et al.* Management of uninvestigated and functional dyspepsia: a Working Party report for the World Congresses of Gastroenterology 1998. Aliment Pharmacol Ther. 1999; 13(9):1135-1148.
- 34. **Talley NJ.** Dyspepsia management in the millennium: the death of test and treat? Gastroenterology. 2002; 122(5):1521-1525.
- 35. **Vaira D,** Vakil N. Blood, urine, stool, breath, money, and Helicobacter pylori. Gut. 2001; 48 (3):287-289.
- 36. **Vaira D,** Vakil N, Menegatti M *et al.* The stool antigen test for detection of *Helicobacter pylori* after eradication therapy. Ann Intern Med. 2002; 136(4):280-287.
- 37. **Canga C,** Vakil N. Upper GI malignancy, uncomplicated dyspepsia, and the age threshold for early endoscopy. Am J Gastroenterol. 2002; 97(3):600-603.
- 38. World Gastroenterology Organization Global Guideline: *Helicobacter pylori* in developing countries. J Clin Gastroenterol.; 45 (5):383-388.